Metabolic Over-Exertion and Resultant Epigenetic Changes to Neurotransmitter Switches Culprit in Febrile and Cannabis-Associated Schizophrenia

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Introduction

The specific mechanism underpinning schizophrenia continues to elude neurologists, although there are a number of important clues which point to a possible culprit.

Abstract

The fact that there is a strong statistical relationship between both high fever during pregnancy and youth cannabis use and schizophrenia is suggestive of neurotransmitter overproduction as the underlying cause.

Both a febrile state and the influence psychotropic drugs share in common that they result in the massive production of brain chemicals which are ordinarily produced only in small quantities. In the case of fever, this overproduction is the result of the elevation in temperature driving a higher rate of production of these chemicals. The chemicals in question, yet to be specifically identified, seem to be produced, ideally, only when a person is having a genuine experience and serve as a basic means of distinguishing between what is the recollection of a memory or a flight of the imagination and what is a real experience. The presence of the chemical or chemicals heightens the emotional gravitas of an experience and in excessive quantities can lead an individual to be unable to distinguish between what is real and what is not (sc. hallucinations.)

Pregnant women and children often experience fevers but do not always develop schizophrenia as a result. There is a genetic component to schizophrenia which is likely associated with a diminished ability to tolerate wide variances in demand for the production of these chemicals and a heightened tendency for epigenetic switches to be permanently altered as a result of transient hyperthermia or drug exposure. Particularly in an individual with a genetic predisposition, either elevated temperature or exposure to psychotropic chemicals may result in a switch telling a cell to produce a specific type of chemical to be stuck in the ‘on’ position, resulting in its continued production at inappropriate times and in inappropriate quantities even after the fever or drug exposure which first triggered the production has long-ceased to be present.

Each instance of the use of a psychotropic drug could be expected to result in comparatively few of these cells to be epigenetically corrupted in this manner. The more instances of use occur, the more cells are modified. When adults are exposed to a fever state or to the same chemical compounds, they tend not to develop schizophrenia, with the latest age for onset of schizophrenia being about 30.

I propose that the reason for the aforementioned phenomenon is that when a brain is still developing, an improperly functioning chemical-producing cell within the brain may be copied during the normal growth process. A brain cell the epigenetic characteristics of which have been modified could be expected to convey the improperly-thrown switches to the newly created cell, thereby multiplying, perhaps by manifold the number of affected cells. Furthermore, epigenetic alterations can be “contagious” and can spread to extant, previously unaffected cells in a youthful brain because the same mechanisms which enhance learning by constantly sending chemical messengers to neighboring cells can convey epigenetic data which may be adopted by the neighboring cells. This as-yet-unrecognized process in which brain cells may cross-reference the epigenetic states of neighboring cells in order to alter their own states is performed with greater frequency in younger brains and is virtually never carried out in aging brains.

Between these two phenomena, the overproduction of “bad trip” chemicals within the brain on an ongoing basis could be explained, as could the tendency of children to be most vulnerable to the fundamental changes which eventually lead to schizophrenia and why schizophrenia worsens with age up until a patient’s twenties.

Conclusion

By identifying the specific epigentic changes responsible for the induction of this condition, the switches may be artificially reverted to a normal state through a gene editing technique in order to halt the production of the chemicals, although reversing the damage done by long-term exposure to these brain chemicals would remain challenging.